The opinion in support of the decision being entered today is *not* binding precedent of the Board.

#### UNITED STATES PATENT AND TRADEMARK OFFICE

# BEFORE THE BOARD OF PATENT APPEALS AND INTERFERENCES

Ex parte BINIE V. LIPPS and FREDERICK W. LIPPS

Appeal 2006-2644 Application 10/047,945 Technology Center 1600

Decided: July 31, 2007

Before TONI R. SCHEINER, ERIC GRIMES, and RICHARD M. LEBOVITZ, *Administrative Patent Judges*.

GRIMES, Administrative Patent Judge.

## DECISION ON REQUEST FOR REHEARING

Appellants have requested rehearing (reconsideration) of the decision entered December 21, 2006. That decision affirmed the Examiner's rejection of claims 9-18 for nonenablement. We have considered Appellants' arguments but decline to change the disposition of the claims in the previous decision. The request for rehearing is denied.

#### DISCUSSION

Appellants argue that the previous decision erred in its interpretation of the "free serum IgE" recited in the claims: "It appears that the decision overlooks or misapprehends the requirement that the method of claim 9 is directed toward the reduction of 'free' IgE. . . . Once bound by appellant's [sic] peptide, the IgE is no longer 'free,' although a reaction product between appellants' peptide and the IgE may be present" (Request for Rehearing ("Req. Rhg.") 2). Appellants point to the Specification's Table 4 as support for their position: "Table 4, column LT-10 of appellants' specification shows declining free-IgE detection as measured in saliva" (Req. Rhg. 3).

We do not agree with Appellants' interpretation of the claim language. We give claims their broadest reasonable interpretation *consistent with the specification*. See In re Morris, 127 F.3d 1048, 1054, 44 USPQ2d 1023, 1027 (Fed. Cir. 1997). Here, the Specification consistently refers to "reducing IgE"; it does not refer to "reducing IgE not bound to LT-10" or the equivalent. For a few examples, see page 1, lines 12-13 ("treatment of ... elevated IgE levels ... to reduce the level"); page 4, lines 2-3 ("reduction in IgE", "reduce elevated IgE level"); page 5, line 10 ("LT-10 lowers IgE level"); and page 6, lines 5-6 ("Both LT-10 and Mono anti-IgE neutralize the circulating IgE and lower the IgE level.").

Appellants' claim interpretation also conflicts with the basis, as we understand it, for the Specification's assertion that lowering IgE levels treats certain disorders. The Specification states:

Our research further revealed that IgE is implicated in (1) Type II diabetes (2) Depression (3) various types of Autoimmune

diseases and (4) Asthma. It was revealed that the level of IgE in patients of these disorders is several times higher than the control normal individuals. . . .

We also found that high levels of IgE caused disruption in the homeostasis of endogenously present other proteins such as nerve growth factor, myoglobin, insulin and Adenosine deaminase. We believe that such disruption in homeostasis for NGF, myoglobin, insulin and ADA may be manifesting the symptoms for these disorders.

(Specification 4: 12-21.) The Specification also states that "in humans oral administration of . . . LT-10 lowers IgE level. We further demonstrated that by lowering the IgE level, other proteins such as NGF, myoglobin, insulin and ADA [are]returned to their normal homeostasis" (*id.* at 5: 9-12).

Thus, as we understand it, the Specification teaches that an elevated level of IgE disrupts the homeostasis of certain other proteins, leading to the symptoms associated with disorders such as asthma and diabetes and that "lowering the IgE level" returns those other proteins to their normal homeostasis. Notably, the Specification does not state that IgE and LT-10 form a complex that no longer interferes with the homeostasis of NGF, myoglobin, etc.; rather, the Specification states that "LT-10 *lowers* IgE level . . . [and] by *lowering* the IgE level, other proteins . . . [are] returned to their normal homeostasis" (emphases added).

Finally, Table 4 does not support Appellants' claim interpretation. Table 4 is headed "IgE levels in saliva" (Specification 15: 1); the Specification concludes that "Glucotrol treatment does not contribute in lowering IgE levels. It is the LT-10 treatment which causes the lowering of IgE" (*id.* at 15: 16-18). The comparison of the Glucotrol and LT-10 results shows that the experiment was not measuring LT-10-bound IgE versus non-

LT-10-bound IgE: since the treatment with Glucotrol did not include administration of LT-10, it could not have resulted in any LT-10-bound IgE.

Appellants also argue that the previous decision improperly raised the issue of whether IgE levels in saliva correspond to those in serum (Req. Rhg. 3). We disagree. The standard for whether an affirmance should be designated a new ground of rejection is whether the appellant has had a "fair opportunity to react to the thrust of the rejection." *In re Kronig*, 539 F.2d 1300, 1302, 190 USPQ 425, 426 (CCPA 1976). The reasoning in our previous decision is in agreement with the Examiner's reasoning, and simply adds one additional factor that supports the Examiner's rejection. To the extent that Appellants had not previously had reason to address the correlation between saliva and serum levels of IgE, they had an opportunity to do so in the Request for Rehearing, and took advantage of it.

To wit, Appellants argue that the "data in the specification qualitatively supports an association between what was measured and what has been claimed" (Req. Rhg. 4). Appellants point to several passages in the Specification, none of which provides a comparison of IgE levels in saliva and in serum.

Appellants also characterize the Specification's "Experiment #3 (page 13, line 20)" as "reasonably show[ing] that the administration of 2 mg/day of LT 10 steadily reduces serum levels of free IgE as measured in saliva over the course of treatment as shown in Table 4" (Req. Rhg. 4).

We do not agree that the data presented on pages 13-15 of the Specification provide the necessary correlation. The Specification states that the data shown in Table 4 represent saliva levels of IgE. See page 13, lines

15-16 ("IgE . . . [was] assayed in saliva") and line 22 ("The results of these experiments are shown in tables 3-7."). Thus, the data in Table 4 represent only *saliva* IgE levels, and do not show any reduction in *serum* IgE levels.

Appellants also argue that the previous decision erred in its treatment of the application's Figure 1 (discussed in the footnote on page 3 of the decision), because pages 18-19 of the Specification explain the meaning of Figure 1's data (Req. Rhg. 4).

Appellants are correct that we previously overlooked the Specification's explanation of the data shown in Figure 1. Figure 1 summarizes the "Day 7" results for each of the treatments shown in Tables 3-7 (Specification 18: 19 to 19: 3). As relevant to this case, Figure 1 presents, in graphic form, some of the data in Table 4. We have considered the data shown in the figure but conclude that it is merely cumulative to those in Table 4; the figure does not contribute substantively to the Specification's disclosure.

Finally, Appellants argue that one of the lines of reasoning in the previous decision is improperly relied on. Specifically, Appellants argue that the lack of data pertaining to peptides smaller than ten amino acids should not be considered relevant because "claim 9 as examined was limited to SEQ ID NO: 2 (Final Rejection, page 2)" (Req. Rhg. 5).

We acknowledge the election of species requirement made by the Examiner (to the 10-mer of SEQ ID NO: 1, not the 15-mer of SEQ ID NO: 2). Because of the election of species, we withdraw our reliance on the reasoning set forth in the second full paragraph on page 6 of the previous decision: since it is unclear from the record that the Examiner examined

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more than the LT-10 embodiment encompassed by the claims, we do not rely on the lack of evidence pertaining to peptides having less than ten amino acids.

#### **SUMMARY**

We modify the previous opinion by (1) withdrawing our reliance on the second full paragraph on page 6, and (2) acknowledging that footnote 2 is in error (and considering Fig. 1). Notwithstanding those modifications, however, we conclude that the instant claims are not enabled by the Specification's disclosure because the evidence of record does not show that administration of LT-10 reduces the level of IgE (as opposed to masking it from anti-IgE binding) or that the measured saliva levels of IgE correspond to the "serum IgE" levels recited in the claims.

### REHEARING DENIED

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